Peat Biomass Smoke Particle Exposure in Rats Decreases Expiratory Time and Increases Left Heart End Diastolic Volume

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Wildland fires, favored by prolonged drought and rising temperatures, generate significant amounts of ambient particulate matter (PM), which has been linked to adverse health outcomes. The eastern North Carolina peat fires of Pocosin Lake in 2008 and Pains Bay in 2011 were some of the more prominent recent wildland fires and were associated with increased cardiovascular hospitalizations. The biological impacts of peat biomass emissions and the specific mechanisms driving these responses are unclear. The purpose of this study was to investigate the cardiopulmonary responses of peat biomass smoke exposure in rats. We hypothesized that PM exposure would dose-dependently alter cardiopulmonary function. Male Sprague-Dawley rats were exposed to 30 µg (Lo PM) or 300 µg (Hi PM) of peat biomass smoke PM extracts suspended in 200 µL of saline, or saline vehicle alone by oropharyngeal aspiration (OA). Immediately following OA rats were placed in a whole-body plethysmograph and ventilatory data were recorded for 12 minutes. One day following OA, rats were anesthetized with isoflurane for ultrasound assessment of cardiovascular function. Hi PM caused decreases in expiratory timing as early as 4-6 minutes after exposure relative to Lo PM ($p = 0.02$) and Vehicle ($p= 0.06$), which resolved shortly thereafter. One day after OA, ultrasounds revealed that Hi PM exposure increased end diastolic volume (EDV) by 16% ($p = 0.03$) over Vehicle and 13% ($p = 0.06$) over Lo PM. In addition, end systolic volume (ESV) and pulmonary artery acceleration/ejection time ratio (PAT/PET) both demonstrated near significant linear trends ($p = 0.07$ for both) with increasing PM dose, both of which may have influenced EDV. Heart rate, isovolumic relaxation time (a lusitrophic factor that could impact EDV), ejection fraction, fractional shortening, and stroke volume were unaffected. These data suggest that exposure to peat biomass may modulate the regulation of ventricular filling and ejection volumes and may alter resistance and pressure gradients across the pulmonary vasculature. Moreover, early pulmonary responses, perhaps triggered by sensory mechanisms, may have precipitated later cardiovascular responses. (This abstract does not reflect USEPA policy)